The daily-practiced post-partum hemorrhage management: an Italian multidisciplinary attended protocol

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Abstract

Postpartum hemorrhage (PPH) is one of the most frequent causes of maternal death and morbidity in the obstetric population globally, causing about a quarter of maternal deaths yearly, and is the leading cause of maternal death worldwide. The management of PPH remains a topic of great debate, even in view of new diagnostic and therapeutic possibilities in recent years, for which, however, the body of evidence available is still scarce, as the standard values are lacking.

The protocol hereby presented was developed after a literature review and during several meetings of an Italian multidisciplinary task group of specialists adopting a modified Delphi method, and is the result of the synthesis of therapeutic operational protocols for the treatment of PPH applied by the different specialties within the team.

This protocol is intended to represent a practical proposal to support clinicians in the management of a particularly complex event that requires the intervention of a multidisciplinary team and the implementation of dedicated management protocols. Clin Ter 2017; 168(5):e307-316. doi: 10.7417/CT.2017.2026

Key words: Blood components, Postpartum hemorrhage (PPH), Protocol, Thromboelastography, Thromboelastometry, Transfusion

Introduction

The World Health Organization defines primary postpartum hemorrhage (PPH) as blood loss greater than or equal to 500 mL within 24 hours of a vaginal delivery. PPH is regarded as severe if the blood loss exceeds 1000 mL; in a Caesarean section, blood loss equal to or greater than 1000 mL can be defined as anomalous (1). Secondary PPH is defined as abnormal bleeding from the genital tract from 24 hours after delivery until 12 weeks postpartum (2,3).

PPH is one of the most frequent causes of mortality and morbidity in the obstetric population globally, causing about a quarter of maternal deaths yearly, and is the leading cause of maternal death worldwide (1). Most deaths occur within 24-48 hours from childbirth (4). According to the latest report of the Centre for Maternal and Child Enquiries (5) on maternal mortality, despite significant improvements in the last 3 years, 66% of deaths due to PPH are still due to “substandard care”(5). PPH is also responsible for 73% of all serious morbidity during pregnancy and is the most common obstetric cause of admission to intensive care units (6).

The estimated frequency is between 5 and 22% of the total deliveries (7). In the last decade, the incidence of PPH in many industrialized countries has increased. For example, the overall rate of PPH in the United States increased by 27.5% from 1995 to 2004 (8, 9).

The causes of PPH can be manifold. In clinical practice, they are summarized as the “4Ts” (10):
- Tone (uterine atony),
- Tissue (placental problems, including retained placenta and abnormal placental implantation),
- Trauma (uterine rupture, cervical laceration, uterine inversion, or birth canal lacerations), and
- Thrombin (in relation to blood coagulation disorders due to thrombin dysfunction).

The main cause of PPH is uterine atony (9). In most cases, the bleeding occurs in the absence of evident risk factors.

The present protocol aims to propose some practical suggestions that could support the clinical management of PPH, in accordance with the knowledge and experience of experts and the various resources available in several cases in Italian hospitals. Moreover, the authors hope that each hospital service will have a massive transfusion protocol to be activated in case of critical hemorrhage with signs of hemodynamic instability and hypoperfusion (1,2,11).

Multidisciplinary hemorrhage protocols for the management of PPH have been recently advocated by many other authors (12-17), as well as professional societies, including the American College of Obstetrics and Gynecology, the UK...
Confidential Enquiry into Maternal and Child Health, the European Society of Anaesthesiology, the French National College of Gynaecologists and Obstetricians, and The Joint Commission in the United States.

Methods

The following protocol was developed after a literature review and during several meetings of a multidisciplinary team of specialists adopting a modified Delphi method; it is the result of the synthesis of therapeutic operational protocols for the treatment of PPH applied by the different specialties within the team. Before the meetings of the experts, an extensive literature search was carried out on PubMed to identify relevant literature. These studies, together with the clinical practice of experts, were the basis for the protocol developed and the suggestions made by the Panel.

In its final version, this protocol obtained the endorsement of the Italian Association of Hospital Obstetricians and Gynecologists (AOGOI), the Association of Italian University Gynecologists (AGUI), the Italian Society of Gynecology and Obstetrics (SIGO), the Confalonieri Ragonese Foundation, and the ANEMO Association.

Management of PPH

The fundamentals of PPH treatment are: 1) maintenance of uterine contractility, obtained by physical or pharmacologic means, 2) maintenance or support of circulation with proper hydration, and 3) prevention or treatment of the established hemorrhagic coagulopathy. Intervention should be made in the “golden hour” to increase the chances of survival of the patient (2,11).

The course of treatment of a patient who experiences PPH requires the close involvement of many professionals: a specialist gynecologist, an anesthetist, a transfusionist, and an interventional radiologist. In a situation in which promptness is vital to reduce any complications in the patient, a multidisciplinary treatment plan aims to help optimize the management of the hemorrhagic event (2,5,11).

The management of PPH is still a topic of great debate, even in view of new diagnostic and therapeutic possibilities in recent years, for which, however, the body of evidence available thus far remains scarce, as are the standard values (such as the use of thromboelastometry and thromboelastography in obstetric and gynecologic settings, early supplementation with fibrinogen, and the composition of transfusion packages).

Protocol structure

The protocol proposed in the present work is based on a pragmatic approach, which allows a subdivision on two points:

Point A is regarding patients with blood losses of between 500 and 1000 mL and no signs of hemodynamic instability, for which basic monitoring measures are provided and the departments involved are alerted.

Point B is regarding patients with blood losses greater than 1000 mL and who are hemodynamically unstable, for which it is suggested that, in addition to the correction of hypoperfusion with fluids, early support of coagulation should be implemented. If available, early support of coagulation should be done by viscoelastic monitoring tools like rotational thromboelastometry (ROTEM) or thromboelastometry (TEM).

It should be highlighted that the suggested measures cannot be carried out in successive order and often need to be implemented simultaneously. In addition to the application of these measures, the knowledge and experience of the individual clinician and the team involved, which can never fail in the specific clinical situation of the patient, and the actual availability of devices and clinical or pharmacologic tools should be considered.

Figures 1 to 4 show the therapeutic measures recommended in this protocol. Unless otherwise specified, the procedures suggested are based on the 2009 Guidelines of the AOGOI (11) and the 2011 Guidelines of the Royal College of Obstetricians and Gynaecologists (2) on postpartum hemorrhage and are in accordance with the therapeutic operational protocols developed at their hospitals by the authors.

It must be made sure that the patient is not suffering from inherited coagulation disorders: prophylaxis and relevant treatment, in case of congenital coagulation dysfunctions, must be agreed with the hematologist who is in charge of the patient, and put in place following the appropriate guidelines. The expert group decided to exclude patients with inherited bleeding disorders from this sum of suggestions.

Point a: blood losses between 500 and 1000 mL with no signs of hemodynamic instability (Fig. 1)

The first action to be taken in case of PPH is to request the cooperation of other medical and paramedic staff, such as a gynecologist and a second expert obstetrician. It is also advisable to alert the anesthetist, as well as the transfusion center (where available), operating room, vascular surgery department, and possibly other nursing staff; where available, the interventional radiology department should also be alerted.

While waiting for support, an assessment of the extent of the bleeding through retroplacental bag, gauze, and drapes is advised. The literature provides evidence that an accurate estimate of blood loss can shorten the transfusion time, thereby reducing the severity of the hemorrhage (12). Obtaining an accurate assessment of blood loss is a well-known problem, and many authors have highlighted the importance of appropriate and periodically repeated training in this regard (13). We are in agreement on this issue, calling for the implementation of a dedicated program of training and continuing education for all obstetrics staff.

Simultaneous with the assessment of blood loss is the need to begin the monitoring of vital signs: blood pressure, respiratory rate, heart rate, electrocardiography (ECG), pulse oximetry, temperature, and diuresis through a urinary catheter. Checking should be done initially every 10 minutes, according to the clinical evolution, and then every 30 minutes. It is useful to record the progress of the vital signs on suitable predesigned graphic sheets, if possible; this allows for fast verification of the data trend, even in an emergency (12). At this stage, a request for blood components should be sent to the transfusion center.
The patient’s two large venous access points (16 G or, better, 14 G) should also be established to clearly separate the route of administration of oxytocics from that of infusions needed to support the blood volume; the use of infusion pumps is preferable. A urinary catheter also needs to be inserted to empty the bladder.

The pharmacologic therapy at this stage includes oxytocin and tranexamic acid (TXA). There is significant variation in practice in this regard. However, a systematic review of all the available uterotonics for the prevention of PPH has found oxytocin to be the first choice, and a recent Cochrane review supported this recommendation (3). According to the Panel, the dose of oxytocin should be increased to switch from prophylaxis to therapy (20 IU in 500 mL saline in 2 hours); if no effect is observed after 20 minutes, a second-line uterotonic may have to be given according to the clinical situation of the patient.

Evidence supporting the early use of TXA in massive hemorrhage has been rapidly increasing. The efficacy of such therapy in decreasing blood loss and transfusion requirements in vaginal and caesarean deliveries has been previously confirmed (12,13). In a recent prospective trial, a significant reduction of secondary measures was found in severe PPH treated with tranexamic acid (18). A recent systematic review with meta-analysis recommended caution in the use of tranexamic acid in the pro-
phyllaxis of postpartum hemorrhage (19). However, current knowledge indicates that the levels of fibrinogen are higher in pregnant than in non pregnant women. The levels increase considerably in the third quarter of pregnancy, in relation to the estrogen levels (20), and then decrease after the expulsion of the placenta (after birth) due to intravascular fibrin deposition, which results in increased consumption of fibrinogen (18). In fact, electron microscopy studies of placental bed tissue biopsies have shown that immediately after a normal birth, an extravascular fibrin “wallpaper” appears on the endometrial surface (21).

According to these mechanisms, TXA, as an antifibrinolytic agent, can be assumed to be an effective therapy to control EPP (18,20). Therapeutic inhibition of fibrinolysis has already proven to be effective in reducing the bleeding in various clinical situations associated with activation and dysregulation of the fibrinolytic system, including cardiac, hepatic, trauma, neurosurgical, and obstetric settings (22). A randomized, multicenter study carried out in 2011 on 144 patients showed that the use of high-dose tranexamic acid (4 g) significantly reduced the amount of blood loss (PHEP defined as blood loss > 800 ml after vaginal delivery), the duration of blood loss, and the need for transfusions and invasive procedures, in the absence of side effects, except for two cases of thrombosis in the group treated with TXA (18). The CRASH 2 study showed that in a trauma patient, the administration of TXA at high doses (loading dose of 1 g during 10 minutes followed by an infusion of 1 g during 8 hours) reduced total-cause mortality from 16% to 14.5% within 4 weeks, with no increase in thrombotic events. Furthermore, this trial showed that the benefit on survival was evident only if the TXA treatment was started within 3 hours after injury, without increasing the risk of cardiovascular occlusive events (23).

The data suggest that TXA should be given at a dose of 30 mg/kg adjusted body weight (ABW). Obese patients can have an excess of 30% fat; thus, the adjusted body weight helps account for the change, assuming 40% of lean mass and 60% of fat mass for anything above the ideal body weight.

At the same time, samples for blood group have to be taken, and repeated blood counts and basic coagulation tests should be carried out: prothrombin time (PT), partial thromboplastin time (PTT), fibrinogen (with the Clauss method), and antithrombin (AT).

AT monitoring is relevant in clinical situations with ongoing bleeding. A decrease in AT may be associated with an increased risk of thrombosis; however, there is no evidence that normal AT levels decrease the thrombotic risks associated with rFVIIa administration. We point out the importance of not given AT during the acute bleeding phase due to the potential risk of worsening coagulopathy (24). Thus far, there are no data available on the stepwise changes in the concentration of AT during the bleeding event. In all likelihood, PPH patients may experience an ongoing decrease comparable with that in trauma patients (24).

Close monitoring is needed to avoid or correct hypothermia and to measure and avoid acidosis (lactates > 2 mmol / L) and desaturation. We suggest that an arterial blood gas analysis be carried out to obtain a baseline hemoglobin level (which is useful for the subsequent monitoring of blood loss), as well as estimation of the arterial oxygen saturation combined with pulse oximetry.

For a correct diagnostic and therapeutic overview of the patient, the source of bleeding should be established by applying the rule of the 4 Ts (10), and the relevant corrective actions should be determined:

TONE (bimanual uterine compression, intracavitary uterine tamponade through a hydrostatic balloon catheter, and use of uterotonics). In the absence of a hydrostatic balloon, a latex glove or a condom can be used with good results, as suggested by the FIGO 2012 Guidelines (25). It should be emphasized that the use of gauze tamponade is now discouraged.

TISSUE (exploration and evacuation of the uterus).

TRAUMA (vaginal lacerations and cervix and/or uterine rupture repair).

THROMBIN (in relation to blood coagulation disorders due to thrombin dysfunction; any coagulation defects should be evaluated and corrected with ROTEM/TEG monitoring, if available).

Point b: blood losses > 1000 ml in patients with hemodynamic instability (Figs. 2-4)

The transition to Point B should be made only when resolution of the effective hemorrhage is not achieved after all the operations referred to in Point A have been put in place. The first procedure referred to in Point B (Fig. 2) is the restoration of the circulating volume by giving the least volume of crystalloids (Ringer-lactate/acetate as the first line) or colloids (26) until the hypoperfusion is corrected, based on an evaluation of the clinical and laboratory variables (sensory state, diuresis, lactates, and deficit base), and hasten the request of blood products. In the administration of colloids, it is crucial to consider the information note issued by the AIFA and the EMA in December 2013 regarding restrictions on the use of medicines containing hydroxyethyl starch (HES) (26).

Then, the path of the protocol is differentiated according to the availability or nonavailability of a POC monitoring tool in the structure that has taken charge of the patient (Figs. 2-4). For both arms of the protocol, the use of warming devices and rapid infusers is appropriate, and the basic conditions should always be ensured: hematocrit > 21%, temperature > 34 °C, pH > 7.20, and Ca++> 1 mmol/L.

In addition to the assessment of coagulation by laboratory standard tests, Point-of-Care (POC) testing should be done, if available. TEG/ROTEM are capable of providing a useful assessment of the coagulation status within minutes. There are numerous case reports of the use of TEG and ROTEM to guide the replacement of blood components in the management of PPH, and studies have confirmed a reduction in blood loss and associated transfusion of blood products with the use of these tests in other settings (13). A strong correlation has been identified between standard coagulation variables and ROTEM variables in women shortly after delivery (27,28).

The FIBTEM is a ROTEM assay that evaluates the fibrinogen component and seems to be well correlated with the standard laboratory fibrinogen levels (29). There is strong evidence that the ROTEM FIBTEM A5 assay can be used as a surrogate for Clauss fibrinogen during PPH. It should be noted that this assay does not measure the same hemostatic
PERFORM ALL ACTIONS PROVIDED IN POINT A

HYPOPERFUSION CORRECTION
Infuse crystalloid (first line) or colloids while waiting for blood
Assess the sensory state, base excess, lactates, urine output

ADMINISTER OXYGEN

TRANSFUSION THERAPY
Always ensure the basic conditions:
- hematocrit > 21%, temperature > 34 °C, pH > 7.20, Ca++ > 1 mmol / L
- If infusion rate > 50 mL / kg / h: use warmers and rapid infusers

Is Point of Care (ROTEM / TEG) monitoring available?

<table>
<thead>
<tr>
<th>NO</th>
<th>YES</th>
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<tbody>
<tr>
<td>&quot;BLIND&quot; TRANSFUSION THERAPY</td>
<td>TRANSFUSION THERAPY GUIDED</td>
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<tr>
<td>While waiting for the laboratory results</td>
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<tr>
<td>- 4 bags of packed red blood cells: 4 units of plasma from a single donor or industrial type</td>
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<tr>
<td>- or 4 bags of packed red blood cells: 2 units of apheresis plasma</td>
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<tr>
<td>- Platelet concentrate, 1 unit of apheresis or buffy coat per 8 bags of packed red blood cells</td>
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<tr>
<td>If PTT or INR &gt; 1.5: transfuse packed red blood cells and plasma (starting dose 20 ml / kg, up to 30 ml / kg for persistent or worsening coagulopathy)</td>
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<tr>
<td>If fibrinogen (Clauss or ROTEM / TEG) ≤ 200 mg / dL, the infusion of the following is recommended:</td>
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<tr>
<td>- fibrinogen concentrate (30-50 mg / kg) or</td>
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<tr>
<td>- cryoprecipitate (1 unit per 10 kg)</td>
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<tr>
<td>TRANSFUSION THERAPY GUIDED</td>
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<tr>
<td>BY ROTEM (see Fig. 3)</td>
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<tr>
<td>OR BY TEG (see Fig. 4)</td>
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PREVENT OUTBREAK OF DIC
Treat the underlying cause (persistent hemorrhage, shock, hypothermia, acidosis) and establish appropriate haemodynamic support

CONSERVATIVE SURGERY AND INTERVENTIONAL RADIOLOGY
Compression sutures (B-Lynch, Hayman), devascularisation (uterine, ovarian or internal iliac arteries), selective embolisation of pelvic vessels

OFF-LABEL USE OF rFVIIa
Bolus of 60-90 µg / kg may be repeated within 15-30 mins

SUBTOTAL OR TOTAL Hysterectomy

DIC = Disseminated intravascular coagulation; INR = International Normalized Ratio; PTT = Partial Thromboplastin Time; rFVIIa = Recombinant factor VIIa

Fig. 2. Protocol: Point B. Management of PPH in case of blood losses > 1000 mL, for a patient with hemodynamic instability
variable that Clauss fibrinogen does but rather provides similar measures of hemostatic competence (14). Some studies have reported that even the TEG-based functional fibrinogen (FF) assay correlates well with the standard von Clauss fibrinogen assay (30,31).

During massive hemorrhage, fibrinogen is one of the first coagulation factors to decrease beyond critical levels. Term pregnant women have an increased concentration of fibrinogen of approximately 4.5-5.8 g/l compared with the normal value of 2.0-4.5 g/l (32). Several recent studies have suggested that fibrinogen is an important predictor of severe PPH (33-37). Some guidelines have indicated that fibrinogen concentrate should be the replacement therapy of choice (38,39); however, evidence from clinical trials is still scarce. Currently, some randomized controlled studies are being done to provide better insight into the early use of fibrinogen concentrate in PPH (40-43).

Coagulation monitoring can be done by using ROTEM or TEG viscoelastic tests. Thus, we present both guided ROTEM therapy (Fig. 3) and guided TEG therapy (Fig. 4). Thus far, no reference values for monitoring by POC testing in obstetric and gynecologic settings have been standardized and shared (28,44,45). Therefore, our suggestions are only indicative to support the decisions of the specialists involved. The information presented herein is not intended to replace the assessment of individual physicians, who base their decisions on the clinical situation of the single patient, according to protocols established by the structure applied and the availability of equipment and blood components.

We consider it appropriate to suggest indications regarding the interpretation of the viscoelastic variables, which may constitute an indicative support in clinical practice.

If POC testing is not available, transfusion in the presence of the effective PPH is done based on clinical indications and not on information obtained from blood chemistry tests (Fig. 2, “blind transfusion therapy”). Therefore, it should be noted that a bag of packed red blood cells contains 280 ml and increases the hematocrit level by 2-3%. When more than 3 or 4 bags have been transfused rapidly, a blood warmer should be used.

There are currently two practices applied in massive hemorrhage: fixed product ratio or individualized procoagulant intervention and factor substitution (39). Patients transfused with a high ratio of red blood cells (RBC) to fresh frozen plasma (FFP) of >1:2 seem to have lower mortality (16,46). In the absence of laboratory information and in case of severe PPH (blood loss > 1000 ml in a hemodynamically unstable patient), we suggest the use of a bag of plasma and platelets for each bag of packed red blood cells while waiting for laboratory values. Regarding the package to be transfused, the following alternative ratios are suggested according to the availability of blood components: 4 bags of packed red blood cells to 4 units of plasma from a single donor or industrial type, or 4 bags of packed red blood cells to 2 units of apheresis plasma. For platelet concentrates, the use of 1 unit by apheresis or buffy coat for every 8 bags of packed red blood cells is suggested.

Emphasis should be given to the suggestive character of the above alternatives, which are not to be considered as recommendations, and the application of which in clinical practice may vary given the uneven distribution and availability of blood components and monitoring equipment.

<table>
<thead>
<tr>
<th>Long EXTEM CT</th>
<th>FFP 20-30 ml/kg</th>
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<tr>
<td>EXTEM ML &gt; 15% →</td>
<td>Normal APTEM → TXA 1 g</td>
</tr>
<tr>
<td>EXTEM A10 &lt; 40 mm →</td>
<td>FIBTEM A5 ≤ 6 mm or A15 ≤ 8 mm → Fibrinogen concentrate 2-4 g</td>
</tr>
<tr>
<td></td>
<td>FIBTEM A5 ≥ 6 mm or A15 ≥ 8 mm → Platelet concentrate</td>
</tr>
<tr>
<td>→ NO RESPONSE</td>
<td>→ GO BACK TO FIGURE 2</td>
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</table>

A10 = Clot firmness (mm) 10 minutes after CT; A5 = Clot firmness (mm) 5 minutes after CT; CT = Clotting time; FFP = Fresh Frozen Plasma; ML = Maximum lysis; TXA = Tranexamic acid

Fig. 3. Protocol: Point B. Transfusion therapy guided by ROTEM

| R > 1 → | Deficiency of coagulation factors → Plasma / Cryoprecipitate |
| R > 0 < 1 → | MA > 54 < 72 → Surgical bleeding → Experienced surgeon |
| MA < 54 → | Functional fibrinogen (FF) → MA > 9 < 29 → Shortage of platelets → Platelets |
|           | MA < 9 → Deficiency of fibrinogen → Fibrinogen |
| → NO RESPONSE | → GO BACK TO FIGURE 2 |

INR = International Normalized Ratio; PTL = Platelets

Fig. 4. Protocol: Point B. Transfusion therapy guided by TEG [rapid TEG (r-TEG)]
It is also desirable that each hospital draw up a massive transfusion protocol to be activated in case of critical hemorrhage with signs of hemodynamic instability and hypoperfusion.

When the results of coagulation tests are available, if the prothrombin time ratio (PTTr) or international normalized ratio (INR) is > 1.5, the Panel suggests infusing plasma, at the same time as the packed red blood cells, at an initial dose of 20 ml/kg and up to a dose of 30 ml/kg in case of persistent or worsening coagulopathy. In cases of altered coagulation, the epidural catheter, which is used during labor, should be left in place until the coagulation level has been normalized.

One of the leading causes of death due to PPH in the Western world is delay in the administration of blood. For example, in a multicenter study carried out in France by Bonnet et al., the time elapsed from the start of PPH to the first transfusion was correlated with the severity of the outcome and mortality: 82 minutes before transfusion for deceased patient compared with a much shorter time for survivors (47).

Considering the rapid decrease of fibrinogen in hemodynamically unstable patients with severe PPH, if the values of fibrinogen, detected by the Clauss method or thrombelastometry/thromboelastography, are less than or equal to 200 mg/dL, we suggest early supplementation with fibrinogen concentrate (30 mg/kg) or cryoprecipitate (1 unit/10 kg of body weight), although this procedure has recently been considered a second-line treatment, with a degree of evidence of 3-I.

Cases that do not respond to the therapies described above require a conservative surgical interventionist approach: balloon tamponade (56); compression sutures (B-Lynch/Hayman), possibly by using “the sandwich effect” (combination of compression sutures with a hydrostatic balloon) (25,48,49) combining devascularization sutures of the uterine, ovarian, and internal iliac arteries; and selective embolization of the pelvic vessels, such as the uterine and cervicovaginal arteries.

The administration of the recombinant activated factor VII (rFVIIa) is somewhat controversial: rFVIIa has been used empirically during obstetric hemorrhage as a last-ditch attempt to prevent hysterectomy, with anecdotal evidence of success (13); however, it is not currently recommended for routine treatment of PPH due to its heavy thrombotic risks (50). If there is no response to the previous measures, the administration of rFVIIa at a dose of a bolus of 60-90 mg/kg can be suggested; this may be repeated within 15-30 minutes as a last resort before hysterectomy. It should be noted, however, that the effectiveness of rFVIIa depends on the values of some basic variables, such as pH, temperature, platelet count (> 50,000/mm³), and fibrinogen level (> 200 mg/dL).

In case of nonresponse to the measures outlined thus far, the application of subtotal or total hysterectomy will be necessary. This decision should be made by the most experienced obstetrician and preferably supported by a second experienced clinician. Subtotal hysterectomy has lower surgical morbidity and is the operation of choice, unless there is trauma to the cervix or lower segment (grade 2-IIb/2-III) (12).

If the cervix and paracolpos are not involved as the source of hemorrhage, subtotal hysterectomy should be adequate to achieve hemostasis, which is the objective of the intervention. Additionally, the procedure is safer, faster, easier to carry out, and less likely to injure the bladder or ureters compared with total hysterectomy (1,51). However, if the lower segment and paracolpos are involved in the hemorrhage, such as in cases of placenta previa, total hysterectomy will be necessary for hemostasis (1,51).

**Conclusion**

There are still many critical points in the management of PPH, starting from the difficulty of properly assessing the extent of the bleeding, and these could dangerously affect the timeliness of diagnosis and corrective and/or therapeutic actions. Delay in initiating appropriate management of PPH is the main factor associated with adverse outcomes: the most important measure at diagnosis of PPH is that immediate action should be taken in the “golden hour” to ensure the best chance of preserving organ function and preventing maternal death.

Another critical point is the involvement of a multidisciplinary team that has the necessary tools to operate at its best and whose members interact competently and promptly. In this sense, the availability of blood products and tools to monitor coagulation is necessary. Unfortunately, because not all hospitals have the same equipment and supplies, this can become a critical issue.

The introduction of thromboelastography and thromboelastometry constitutes an important innovation in the diagnosis of coagulopathy and the management of blood transfusion in cardiac, liver, and trauma settings. The relevant literature shows a good connection between the results of thromboelastometry/thromboelastography and those of the traditional coagulation tests, and in non-gynecologic settings, POC monitoring is a rather common practice (52-55). However, data on the use of POC monitoring in obstetric and gynecologic emergencies are still limited and therefore require further study and analysis (28,44,45).

The authors developed the protocol presented here because of the complex situations they encounter in their daily clinical practice, for which the evidence is scarce and expert opinions are relied on. The importance of this protocol lies in the practical information suggested, which can support clinicians in addressing problems related to PPH in clinical practice. Moreover, the authors hope to stimulate debate and direct attention to the need to develop dedicated protocols for PPH management.

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Dr. Messiha declares a financial relationship with CSL Behring, Bayer, Novo Nordisk, Baxter, Pfizer.

Dr. Agostini declares a financial relationship with Novo Nordisk.

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